

AMENDMENTS TO THE CLAIMS

The following listing of the claims includes the amendments made in the Amendment and Response After Final mailed on September 24, 2007.

Please amend claims 71 and 113.

Please add new claims 114-137.

1-53. **(Canceled)**

54. **(Previously Presented)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated autoimmune disorder comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT- β -R blocking agent comprises a soluble LT- β -R.

55-56. **(Canceled)**

57. **(Previously Presented)** The method according to claim 54, wherein the subject is a human.

58. **(Previously Presented)** The method according to claim 54, wherein the LT- β -R blocking agent comprises a soluble LT- β -R having a ligand binding domain that can selectively bind to a surface LT ligand.

59. **(Previously Presented)** The method according to claim 54, wherein the LT- β -R blocking agent comprises a soluble LT- β -R fused to one or more heterologous protein domains.

60. **(Previously Presented)** The method according to claim 111, wherein the LT- β -R blocking agent comprises a monoclonal antibody directed against LT- β -R.

61-65. **(Canceled)**

66. **(Previously Presented)** The method according to claim 58, wherein the soluble LT- β -R is administered in an amount sufficient to coat LT- β ligand -positive cells for 1 to 14 days.
67. **(Canceled)**
68. **(Previously Presented)** The method according to claim 59, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
69. **(Previously Presented)** The method according to claim 59, wherein the pharmaceutical composition is administered to the subject via oral administration.
70. **(Previously Presented)** The method according to claim 59, wherein the pharmaceutical composition is administered to the subject via parenteral administration.
71. **(Currently amended)** A method for inhibiting lymphotoxin- β receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated autoimmune disorder comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent comprising a soluble LT- β -R fused to one or more heterologous protein domains, and a pharmaceutically acceptable carrier, wherein the ~~LT- β -R blocking agent~~ comprises a soluble LT- β -R fused to one or more heterologous protein domains has a ligand binding domain that can selectively bind to a surface LT ligand.
72. **(Previously Presented)** The method according to claim 71, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
73. **(Previously Presented)** The method according to claim 71, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.

74. **(Previously Presented)** The method according to claim 73, wherein the heterologous domain further comprises a human immunoglobulin Fc domain.
75. **(Previously Presented)** The method according to claim 74, wherein the composition is administered to the subject at a dose of about 1 mg/kg.
76. **(Previously Presented)** The method according to claim 74, wherein the composition is administered to the subject via oral administration.
77. **(Previously Presented)** The method according to claim 74, wherein the composition is administered to the subject via parenteral administration.
78. **(Canceled)**
79. **(Previously Presented)** The method according to claim 71, wherein the autoimmune disorder is selected from the group consisting of psoriasis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
80. **(Previously Presented)** The method according to claim 100, wherein the chronic inflammatory disorder is inflammatory bowel disease.
81. **(Previously Presented)** A method for inhibiting lymphotoxin- β receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated autoimmune disorder comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent comprising a soluble LT- β -R fused to a heterologous domain comprising a human immunoglobulin Fc domain, and a pharmaceutically acceptable carrier, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.
82. **(Previously Presented)** The method according to claim 81, wherein the composition is administered to the subject at a dose of about 1 mg/kg.

83. **(Previously Presented)** The method according to claim 81, wherein the composition is administered to the subject via oral administration.
84. **(Previously Presented)** The method according to claim 81, wherein the composition is administered via parenteral administration.
85. **(Canceled)**
86. **(Previously Presented)** The method according to claim 81, wherein the autoimmune disorder is selected from the group consisting of psoriasis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
87. **(Previously Presented)** The method according to claim 80, wherein the chronic inflammatory bowel disease is Crohn's disease or ulcerative colitis.
88. **(Previously Presented)** The method according to claim 59, wherein the heterologous protein domain further comprises a human immunoglobulin Fc domain.
89. **(Previously Presented)** The method according to claim 54, wherein the autoimmune disorder is selected from the group consisting of psoriasis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, and uveitis.
90. **(Previously Presented)** The method according to claim 113, wherein the chronic inflammatory disorder is selected from the group consisting of inflammatory bowel disease is Crohn's disease or ulcerative colitis.
91. **(Previously Presented)** The method according to claim 54, wherein the autoimmune disorder is rheumatoid arthritis.

92. **(Previously Presented)** The method according to claim 70, wherein the parenteral administration is subcutaneous.
93. **(Previously Presented)** The method according to claim 70, wherein the parenteral administration is intravenous.
94. **(Previously Presented)** The method according to claim 70, wherein the parenteral administration is intralesional.
95. **(Previously Presented)** The method according to claim 71, wherein the autoimmune disorder is rheumatoid arthritis.
96. **(Previously Presented)** The method according to claim 84, wherein the parenteral administration is subcutaneous.
97. **(Previously Presented)** The method according to claim 84, wherein the parenteral administration is intravenous.
98. **(Previously Presented)** The method according to claim 84, wherein the parenteral administration is intralesional.
99. **(Previously Presented)** The method according to claim 81, wherein the autoimmune disorder is rheumatoid arthritis.
100. **(Previously Presented)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated chronic inflammatory disease comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT- β -R blocking agent comprises a soluble LT- β -R.

101. **(Previously Presented)** The method according to claim 100, wherein the LT- β -R blocking agent comprises a soluble LT- β -R having a ligand binding domain that can selectively bind to a surface LT ligand.
102. **(Previously Presented)** The method according to claim 100, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.
103. **(Previously Presented)** The method according to claim 100, wherein the LT- β -R blocking agent comprises a soluble LT- β -R fused to one or more heterologous protein domains.
104. **(Previously Presented)** The method according to claim 103, wherein the heterologous protein domain comprises a human immunoglobulin Fc domain.
105. **(Previously Presented)** The method according to claim 100, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
106. **(Previously Presented)** The method according to claim 100, wherein the pharmaceutical composition is administered to the subject via oral administration.
107. **(Previously Presented)** The method according to claim 100, wherein the pharmaceutical composition is administered to the subject via parenteral administration.
108. **(Previously Presented)** The method according to claim 107, wherein the parenteral administration is subcutaneous.
109. **(Previously Presented)** The method according to claim 107, wherein the parenteral administration is intravenous.
110. **(Previously Presented)** The method according to claim 107, wherein the parenteral administration is intralesional.

111. **(Previously Presented)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a subject having a Th1 cell-mediated autoimmune disorder or a Th1 cell-mediated chronic inflammatory disease comprising administering to the subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent, and a pharmaceutically acceptable carrier, wherein the LT- β -R blocking agent comprises an antibody directed against LT- β -R.

112. **(Previously Presented)** The method according to claim 111, wherein the autoimmune disorder is selected from the group consisting of psoriasis, diabetes mellitus, multiple sclerosis, sympathetic ophthalmia, rheumatoid arthritis, and uveitis.

113. **(Currently amended)** The method according to claim 111 ~~90~~, wherein the chronic inflammatory disease is inflammatory bowel disease.

114. **(New)** The method according to claim 71, wherein the subject is a human.

115. **(New)** The method according to claim 81, wherein the subject is a human.

116. **(New)** The method according to claim 100, wherein the subject is a human.

117. **(New)** The method according to claim 77, wherein the parenteral administration is subcutaneous.

118. **(New)** The method according to claim 77, wherein the parenteral administration is intravenous.

119. **(New)** The method according to claim 77, wherein the parenteral administration is intralesional.

120. **(New)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a human subject having rheumatoid arthritis comprising administering to the human subject a

pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent comprising a soluble LT- β -R fused to a heterologous domain comprising a human immunoglobulin Fc domain, and a pharmaceutically acceptable carrier, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.

121. **(New)** The method of claim 120, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.

122. **(New)** The method of claim 120, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.

123. **(New)** The method of claim 120, wherein the pharmaceutical composition is administered to the subject via subcutaneous administration.

124. **(New)** The method of claim 123, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.

125. **(New)** The method of claim 123, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.

126. **(New)** A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a human subject having ulcerative colitis comprising administering to the human subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent comprising a soluble LT- β -R fused to a heterologous domain comprising a human immunoglobulin Fc domain, and a pharmaceutically acceptable carrier, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.

127. **(New)** The method of claim 126, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.

128. (New) The method of claim 126, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.
129. (New) The method of claim 126, wherein the pharmaceutical composition is administered to the subject via subcutaneous administration.
130. (New) The method of claim 129, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
131. (New) The method of claim 130, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.
132. (New) A method for inhibiting lymphotoxin- β -receptor (LT- β -R) signaling in a human subject having Crohn's disease comprising administering to the human subject a pharmaceutical composition comprising an effective amount of an LT- β -R blocking agent comprising a soluble LT- β -R fused to a heterologous domain comprising a human immunoglobulin Fc domain, and a pharmaceutically acceptable carrier, wherein the soluble LT- β -R comprises a functional sequence of amino acids selected from the amino acids of SEQ ID NO: 1, wherein the functional sequence comprises the LT- β -R ligand binding domain.
133. (New) The method of claim 132, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.
134. (New) The method of claim 132, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.
135. (New) The method of claim 132, wherein the pharmaceutical composition is administered to the subject via subcutaneous administration.

136. (New) The method of claim 135, wherein the pharmaceutical composition is administered to the subject at a dose of about 1 mg/kg.

137. (New) The method of claim 135, wherein the pharmaceutical composition is administered to the subject at a dose of 1 mg/kg to 5 mg/kg.